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Pathology

Study Outlines

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Suffixes & their Different Meanings

gia - pain
emia - condition of Blood
ogue - Carry away
ka - an attack
gia - pain
lle - hernia or protrusion
ectomy - to cut
Graph - to write
Itis - inflammation
Logy - discourse or disease
Malacia - softening
Mania - madness
Odynia - pain
Oid - form
Oma - tumor or swelling
Opia - condition of eye
Pathy - relating to disease
Phobia - fear
Plasty - to form
Raphy - suture or seam
Rhagia - burst forth
Rhea - to flow.
Scopia - to see
Tomy - to cut
Uria - refers to urine

Pseudo - false
Poly - many
Pro - before
- to pus
- fever
- backward or behind
or Syn - together or

Medical Prefixes

A or An - same as un or in
Ana - against - up - thru
Ant or anti - " or opposed
Apo - from
Bi - two
Contra - against
Dia - thru
Dys - defective or difficult
Ec - Ecto - Ex - away from
Epi - upon or over
Extra - outside
Entero - intestine
Gastro - stomach
Haemo - refers to blood
Hemi - half
Hetero - abnormal
Hydro - water
Hyper - above
Infra - below
Inter - between
Intra - inside
Leuco - white
Litho - relating to stone
Luetic - Venereal or Syphilis
Micro - invisible
Melano - pigmented
Meso - middle
Mon - single
Multi - many
Myelo - brain & Spinal Cord
Myo - muscle
Neuro - nerve
Odonto - teeth
Oligo - lack of
Osteo - bone
Para - thru or near
Peri - around

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CHAPTER I.

DEFINITIONS.

I. PATHOLOGY.
The science that treats of disease in all its aspects.

II. ETIOLOGY.
The science that treats of the causes of disease.

III. PROGNOSIS.
Advance judgment regarding the cause, duration and termination of a disease.

IV. DIAGNOSIS.
The recognition of a disease from its symptoms.

V. ANATOMIC DIAGNOSIS.
This is divided into two parts:-

A. Ante mortem.
That which is made before death.

B. Post mortem.
That which is made after death.

VI. DIFFERENTIAL DIAGNOSIS.
By comparing one disease with other diseases.

VII. EXCLUSION DIAGNOSIS.
By excluding all other known conditions.

VIII. PHYSICAL DIAGNOSIS.
This is accomplished by questioning, inspection, palpation, touching and feeling.

A. Percussion.
Tapping and listening to the different sounds.

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B. Auscultation.

Listening to the sounds in a given area, with or without, the aid of a stethoscope.

IX. LABORATORY DIAGNOSIS.

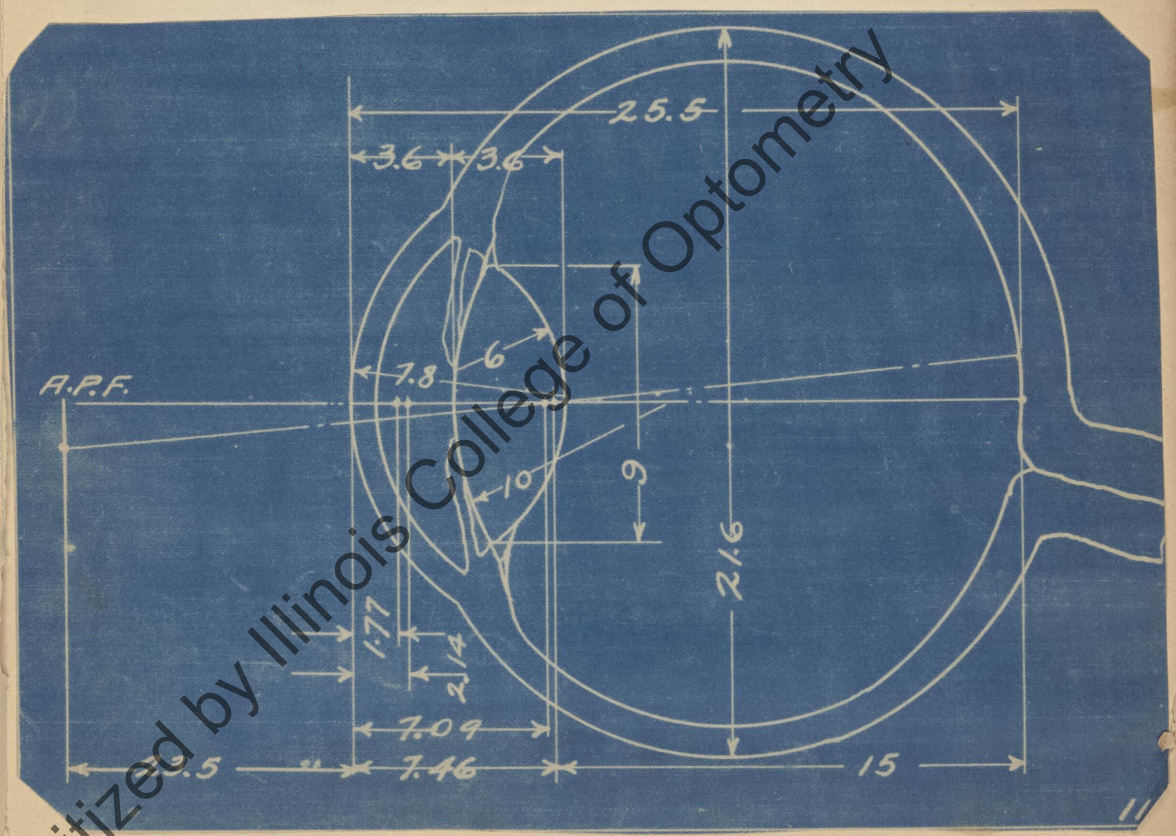
All means by which the fluids, tissues and organs that make up the body are examined, other than those mentioned in physical diagnosis.

X. TOPOGRAPHIC DIAGNOSIS.

Based on the seat of the lesion (any diseased area).

XI. THERAPEUTICS.

All means used in the treatment of diseases.



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CHAPTER II.

INFLAMMATION.

I. DEFINITION.

This is a protective response of body tissue to irritation and is characterized by redness, swelling, heat, pain, impaired function, and as a rule, a discharge.

II. STAGES OF INFLAMMATION.

A. Irritation: Dilation of the capillaries.

Passive / active

B. Hyperemia: Increased blood supply causing redness.

C. Congestion: Abnormal amount of blood in a part.

D. Stasis: In which there is blood neither coming in nor going out.

E. Migration: A passing of white blood corpuscles through the capillary walls to the affected area.

F. Diapedesis: In which the red blood corpuscles break through the vessel walls into the affected area.

G. Exudation: In which there is a discharge of blood serum, or pus, or both.

III. CAUSES OF INFLAMMATION.

A. Infection: when caused by some invading organism such as bacteria.

B. Non-infectious: when due to some foreign inanimate body or by pressure.

C. Idiopathic: when the cause is unknown.

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IV. GENERAL SYMPTOMS OF INFLAMMATION.

A. Redness.
B. Swelling.
C. Tenderness. HEAT
D. Pain.
E. Impaired function.
F. Destruction of terminal nerve endings.

V. RESULT OF INFLAMMATION.

A. Resolution: the white blood corpuscles destroy the invaders without discharge of pus.

B. Suppuration: during the process many white blood corpuscles are destroyed by the bacteria. This results in the destruction of other tissue cells in the area. This mass becomes mixed with blood serum and forms a material called pus.

VI. TREATMENT OF INFLAMMATION.

A. Prophylactic: any means used to prevent the development or spread of disease.

B. Medical.

C. Surgical.

D. Mechanical.

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CHAPTER III.

OCULAR INSPECTION.

I. THINGS TO BE NOTICED BY SIMPLE INSPECTION OF THE EYE.

A. Are the eyelids red?
B. Are the margins of the lids red?
C. Are the lashes stuck together, singly or in a group?
D. Are the lids stuck together?
E. Is there an excessive flow of tears?
F. Are there any signs of styes?
G. Are there any lumps or kernels on the lids?
H. Is the cornea clear or hazy?
I. Are there any spots on the cornea?
J. Do the eyelashes rub the eyeball?
K. Do the eyelids turn out or in?
L. Do the eyelids droop?
M. Is there any growth on the eyeball?
N. Is there any photophobia?
O. Are there any foreign bodies in the eye?

II. OBJECTIVE METHODS OF INVESTIGATION.

A. Keratoscopy.
B. Ophthalmoscopy.
C. Focal or oblique illumination.

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CHAPTER IV.

EXAMINATION OF THE EYE AND ITS APPENDAGES.

I. LACRIMAL APPARATUS.

A. Palpate the lacrimal gland for enlargement, prolapse or tumor.

B. With the patient's eyeball turned toward the nose, press the finger over the lacrimal sac and observe the puncta to determine the presence of pus.

II. LIDS.

A. Note whether ptosis is present or not.

B. Examine for oedema, swelling or redness.

C. Examine the margins for trichiasis, redness, swelling, discharge, scales, crusts, ulcers, tumors etc.

D. Examine the conjunctiva for congestion, thickening, granulations or discoloration.

III. CONJUNCTIVA.

A. Examine the ocular conjunctiva for congestion, thickening, chemosis and tumors.

B. Conjunctival discharges are classified as:

1. Watery: tears, epiphora.
2. Mucous: mucilaginous but clear.
3. Mucopurulent: tenacious, white or yellow.
4. Purulent: creamy, running out of the eyes when the lids are separated.

IV. CONGESTION OF THE EYEBALL.

A. Conjunctival: vessels are movable with the conjunc-

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tiva and fade as they approach the cornea.

B. Ciliary (or circumcorneal): a fine vessel congestion, most intense around the cornea; and pink or violet in color. The vessels do not move with the conjunctiva.

C. Scleral: conjunctiva movable over it. May be a localized congestion of the fine blood vessels or general congestion of the large blood vessels, perforating the sclera.

V. SCLERA.

Examine for localized swelling, congestion, bulging, or areas of discoloration.

VI. OBLIQUE ILLUMINATION.

A. Use a dark room.

B. Hold a +20D, or stronger, so that the image of the light is focussed upon the eye.

C. Another +20D lens may be used to examine the area illuminated.

VII. CORNEA.

A. Anterior surface.

1. Examine by oblique illumination for irregularities, blood vessels, foreign bodies, blisters, ulcers, depressions and opacities.

2. The reflection of a window on the cornea, may, when the patient moves his eyes, reveal distorsions.

B. Deep layers.

Examine for opacities, leukoma and macula by day-light; and for nebula by oblique illumination.

—— = dense
~~~ = shallow } opacities
~~~ = deep

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C. Aqueous chamber.

1. Note whether shallow or not - i.e., the distance between the posterior surface of the cornea and the anterior surface of the iris and lens.

2. Note the clearness of the aqueous, and also if pus and exudate (hypopyon) or blood (hyphaemia) are present.

VIII. IRIS.

A. Compare the iris of one eye with that of the other.

B. Muddiness from congestion causes a loss of the fine markings on the surface.

C. Masses of exudations, tumors or pigment-spots may be observed.

D. If the lens is absent or dislocated, quivering of the iris may be seen.

IX. PUPIL.

A. Note if it is in the center of the iris; and notice its shape.

B. Note reaction to light, by throwing light, by oblique illumination, in and out of the eye.

X. LENS.

A. Examine by direct and oblique illumination.

B. Note opacities or any displacement.

C. A moderate amount of haze, sometimes quite brownish, may be normally present in advanced age, with useful vision present.

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XI. VITREOUS HUMOR.

Examine for exudate, by direct or oblique illumination, and also with the ophthalmoscope.

XII. ORBIT.

Palpate the bony edges of the orbit for irregularities.

XIII. EYEBALL.

A. Note position, as exophthalmos, enophthalmos or irregularity in size.

B. Tension. ——— = Protrusion ~~~~~ = sunken eyeball.

1. Have patient look down: with first two fingers press thru the upper lid until the eyeball is felt.

2. By alternating the pressure of the two fingers the sense of fluctuation is used to determine the tension.

3. The degree of tension may be expressed thus:

T. + 1 = somewhat higher than normal in tension.

T. + 2 = decided rise in tension.

T. + 3 = stony hardness.

T. - 1 = somewhat softer than normal.

T. - 2 = decidedly softer than normal.

T. - 3 = very soft.

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CHAPTER V.

DISEASES OF THE LACRIMAL APPARATUS.

I. SECRETING PORTION.

A. Lacrimal gland is rarely affected.

B. The following diseases affect it:

1. Acute non-suppurative inflammation (dacryoadenitis)
2. Acute suppuration.
3. Chronic inflammation (causing hypertrophy).
4. Cystic distention of the ducts (dacryops)
5. Tumors, tuberculosis and syphilis.

II. CONDUCTING PORTION.

A. Puncta and Canaliculi: may show congenital malposition or stenosis (atresia). The most common condition is displacement of the puncta due to ectropion. Foreign bodies and wounds may close the lumen.

Symptom: Epiphora (lacrimation) especially in windy and cold weather.

III. DISEASES OF THE LACRIMAL SAC.

A. Chronic Dacryocystitis.

1. Synonym.
Blennorrhea of the lacrimal sac.

2. Definition.
Chronic catarrhal inflammation of the mucous lining of the lacrimal sac.

3. Etiology.
Structure of the nasal duct due to nasal diseases, trauma of the bone, and polypi.

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4. Subjective symptoms.
   Epiphora and subjective symptoms of chronic conjunctivitis.

5. Objective symptoms.
   Sac is distended and is felt as an elastic tumor. When pressed, the contents are forced out thru the puncta or nose. Lids are red and swollen. There may be conjunctival discharge. Conjunctivitis and blepharitis are present. The caruncle is swollen.

6. Course.
   May exist for years, but often becomes abscessed. The disease as a rule does not improve. Necrosis of adjoining bone tissue may result.

7. Diagnosis.
   Lacrimation, and discharge of muco-purulent fluid thru puncta, when pressure over sac is exerted, is conclusive.

8. Treatment.
   Medical and Surgical.

B. Abscess of the Lacrimal Sac.

1. Synonyms.
   Purulent dacryocystitis; Phlegmon.

2. Definition.
   Acute suppurative inflammation of the sac and surrounding tissues.

3. Etiology.
   Follows chronic dacryocystitis. Infection is the immediate cause.

4. Pathology.
   Pyogenic bacteria excite an acute inflammation.

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5. Subjective Symptoms.

Sudden attack of severe and throbbing pain in the region of the sac.

6. Objective Symptoms.

Skin is tense, swollen, red and tender. Lids are swollen. General fever, anorexia and lassitude may be present. Unless opened the abscess may burst on the surface and leave a permanent fistulous tract.

7. Diagnosis.

By the history of lacrimation; and the involvement of the sac by abscess formation. Must not be confused with abscess of the lids, at the inner canthus, or bone diseases, in that region.

8. Treatment.

Medical and surgical.

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CHAPTER VI.

DISEASES OF THE LIDS.

I. BLEPHARITIS MARGINALIS. Results from Conjunctivitis.

A. Synonyms.

Blepharitis ciliaris; Tinea tarsi; Blepharoadenitis.

B. Definition.

A chronic inflammation of the margins of the lids, accompanied by the formation of scales and crusts, congestion, thickening and ulceration.

C. Etiology.

Underlying cause is a badly treated conjunctivitis. Error of refraction, poor hygiene, lowered general health, late hours, dust and smoke, are also contributory causes.

D. Clinical Classification.

1. Ulcerative.

In which lashes fall out but do not grow again.

2. Non-ulcerative.

In which lashes fall out and are replaced.

E. Symptoms.

General eye symptoms. At first, margins are hyperemic; which condition comes and goes, when there is exposure to wind, dust, smoke etc. or when the eyes are strained or late hours are kept. Later there is a formation of scales and crusts (non-ulcerative type), or minute pustules are found, which rupture and form hard crusts and scabs, under which ulcerations occur (ulcerative type).

F. Course.

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Most common in children. May last for years causing a loss of lashes and thickened and everted lids.

G. Treatment.

Mechanical, medical (local and constitutional) and correction of hygienic errors.

II. HORDEOLUM.

A. Synonym.

Stye.

B. Definition.

An acute, circumscribed, suppurative inflammation at the margin of the lids, having its beginning in the glands.

C. Etiology.

Eyestrain, impaired vitality, digestive disturbances. The immediate cause is a pyogenic infection.

Pus formation

D. Symptoms.

At first, a burning and itching followed by a red, swollen area at the lid margin. An abscess forms, comes to a head, and finally discharges. An extensive oedema may be present.

E. Course.

Three days to a week, or more often, they are repeated. Occasionally they do not reach the stage of suppuration, but are aborted, or remain as a hard swelling (blind stye).

F. Treatment.

Medical (local and systemic); thermal; surgical; and mechanical.

III. CHALAZION.

A. Synonyms.

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Tarsal tumor; Meibomian cyst.

B. Definition.

Chronic affection of the meibomian glands with a hard swelling in the lids.

C. Etiology.

Chalazia may be due to infection. Most cases are due to the stoppage of a Meibomian duct and an accumulation of the discharge in a gland.

D. Pathology.

Chronic inflammation, showing formation of granulation tissue, originating in the gland. Microscopically the tumor shows little connective tissue, but many cells and no capsule.

E. Subjective Symptoms.

Occasionally there are some inflammatory symptoms in the beginning. The roughened conjunctiva may cause irritation.

F. Objective Symptoms.

Appears as a round or elongated tumor, size of a pea to a walnut. It firmly adheres to the tarsal plate and the skin is movable over it. The conjunctiva over it is dark in color.

G. Course.

May appear without inflammation or at first it may be mistaken for a stye. In the later stages the center of the chalazion may break down and discharge a yellow fluid on the conjunctiva. This is followed by the formation of polypoid masses. Rue Chalazia may be single or multiple. They may disappear spontaneously or their course may be protracted.

H. Treatment.

Thermal, Medical and Surgical.

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IV. ENTROPION.

A. Definition.

A condition in which the lid margin is turned in toward the eyeball.

B. Etiology.

Commonly due to scar contraction of the palpebral conjunctiva (cicatricial entropion) which is due to trachoma, burns or wounds. It may be caused by spasmodic condition of the Orbicularis muscle of the lower lid as seen in elderly people (spasmodic entropion). The spasmodic type is also seen in children, when great photophobia is present, as found in ulcers of the cornea.

C. Symptoms.

Lashes brush against the eyeball, eventually causing irritation and congestion of the eyeball and corneal ulceration.

D. Treatment.

Surgical.

V. ECTROPION.

A. Definition.

A turning outward of the lid margin.

B. Etiology.

Due to thickening of the conjunctiva and margin of the lid; to scars on the skin due to wounds, burns, ulceration or caries of the orbit; to relaxation of the tissues of the lids or paralysis of the Orbicularis muscle. It occurs mostly in elderly people.

C. Symptoms.

Conjunctiva is exposed. Epiphora, irritation and chronic conjunctivitis are present. The lower lid is more often affected.

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D. Treatment.
Medical and surgical.

VI. PTOSIS.

A. Definition.
A drooping of the upper lid.

B. Etiology.
A paralysis or insufficiency of the levator palpebrarum muscle. It may be congenital or acquired. The latter form may be associated with paralysis of other muscles supplied by the third nerve, following diseases such as syphilis or brain diseases. It may also be due to mechanical causes, as in the increased size of the lid when it is affected by trachoma, tumors etc.

C. Symptom.
The patient tries to raise the lid by holding his head back and contracting the frontalis muscle.

D. Treatment.
Surgical.

VII. INJURIES OF THE LIDS.
Wounds and burns may be followed by ectropion, entropion and symblepharon.

VIII. MISCELLANEOUS DISEASES OF THE LIDS.

A. Emphysema.
Inflation of the subcutaneous tissue by air. Appearance of oedema; but a crackling sensation is experienced when palpated.

B. Ecchymosis (Black Eye).
Settling of blood in the loose subcutaneous tissue following a contusion.

C. Oedema.

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Serous exudate into the subcutaneous tissue causing fluctuating swelling. Follows injuries, disease of the lids, urticaria (hives), nephritis, and cardiac diseases. It may be idiopathic.

D. Abscess of the lid.
Usually traumatic.

E. Syphilis.
Rare. May occur as a chancre, mucous patch of the conjunctiva, gumma (nodular tumor), or tertiary ulcer.

F. Lupus.
Tubercular disease of the skin.

G. Blepharospasm.
Involuntary contraction of the orbicularis muscle. May be a tonic or clonic spasm. Fibrillary twitching is the most common form. May be hysteric in origin. It may appear as a symptom of some disease.

H. Lagophthalmos.
Lids cannot be completely closed. Due to injuries, scar contractions, exophthalmos, and to paralysis of the orbicularis muscle.

I. Diseases of the skin of the lids.

1. Erythema (redness due to capillary congestion).
2. Eczema (inflammation with vesicles, infiltration, watery discharges, and scales and crusts).
3. Erysipelas herpes zoster (inflammation with small vesicles in clusters).
4. Syphilis.

J. Epicanthus.
Congenital deformity. A ridge of skin extends from the inner end of the eyebrow to the side of the nose, causing the latter to have the appear-

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ance of a broad bridge.

K. Coloboma of the lid.
Cleft in the eyelid.

IX. TUMORS OF THE LID.

A. Benign.
Warts, dermoid cysts, milia, cutaneous horns, small transparent cysts at lid margins (glands of Moll) Molluscum contagiosum, Xanthelasma and vascular tumors.

B. Malignant.
Sarcoma, (rare). Rodent ulcers (carcinomata).

= = tumor small x size of pin head.

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CHAPTER VII.

CONJUNCTIVITIS (OPHTHALMIA).

I. CLASSIFICATION.

- Conjunctivitis
  - Catarrhal
    - Acute
    - Chronic
  - Purulent
    - Ophthalmia neonatorum
    - Gonorrheal conjunctivitis
  - Follicular
  - Granular - Trachoma
  - Phlyctenular
  - Membranous
    - Croupous
    - Diphtheritic

II. IMPORTANT NOTE.

The examiner should learn to distinguish between the varieties of conjunctival discharge.

A. Watery (tears): Occurs in stenosis of the conducting apparatus etc.

B. Mucous: Clear and mucilaginous. Example: Chronic conjunctivitis.

C. Mucopurulent: White or yellow and tenacious. Example: Acute and chronic conjunctivitis.

D. Purulent: Creamy. Runs out of the eye when lids are separated. Example: Gonorrheal conjunctivitis.

III. ACUTE CATARRHAL CONJUNCTIVITIS.

A. Synonyms.

Acute mucopurulent, or acute contagious conjunctivitis; and acute epidemic conjunctivitis, or "Pink-eye."

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B. Definition.

An acute catarrhal inflammation, particularly of the conjunctiva of the lid.

C. Etiology.

Exposure to wind, smoke, dust, irritating gases or foreign bodies. Infections are due to Bacillus Koch-Weeks, Bacillus Morax-Axenfeld, pneumocccci, staphylococci and streptococci. It is epidemic in Spring and Fall.

D. Clinical Varieties.

Simple and infectious; the latter usually due to bacillus Koch-Weeks.

E. Subjective Symptoms.

About 36 hours after exposure, patient complains of stiffness of the lids, burning, smarting, photophobia, and a sensation of foreign bodies. No actual pain, but great discomfort, especially in the evening.

F. Objective Symptoms.

Lids swollen and red. Bulbar conjunctiva congested, that of lids thick, rough and congested. There is a mucopurulent discharge at the root of the lashes or on the conjunctiva. The lids stick together in the morning. Lacrimation, and maybe, a slight interference with vision; but mucous discharge on the cornea. There may be minute ulceration of the cornea. The eye appears red; hence, the term "Pink-eye".

G. Diagnosis.

Made by the muco-purulent discharge, deep conjunctival congestion, clear vision and absence of pain. In the true "Pink-eye", there is a history of contagion.

H. Course.

As a rule, one eye is attacked a few days in

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advance of the other. Most cases recover quickly. The stage of discharge may last a week or more. Chronic conjunctivitis or blepharitis may be the sequelae.

I. Treatment.

Prophylactic and medical.

IV. CHRONIC CATARRHAL CONJUNCTIVITIS.

A. Definition.

A chronic catarrhal inflammation usually affecting the palpebral conjunctiva only.

B. Etiology.

Dust, night work, late hours, error of refraction, nasal catarrh, and constitutional diseases such as rheumatism and gout. May follow acute conjunctivitis.

C. Subjective Symptoms.

Smarting, itching, burning, feeling of foreign bodies, blurring, photophobia, dryness, heaviness and sleepiness.

D. Objective Symptoms.

The conjunctival appearance varies from slightly red to deep congestion. There may be cheesy deposits on the surface. The lid margins are hyperemic, with a watery or purulent discharge. The skin is excoriated at the outer and inner canthi. The discharge sticks the lids together in the morning.

E. Course.

Runs a long course with increase of symptoms at intervals. Both eyes are usually affected.

F. Treatment.

Medical (local and systemic); and mechanical. Sometimes the disease is intractable.

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V. OPHTHALMIA NEONATORUM.

A. Synonyms.

Purulent conjunctivitis in the infant; acute blennorrhea; gonorrheal conjunctivitis in the newborn.

B. Definition.

Severe purulent conjunctivitis of the newborn due to infection by the gonococcus of Neisser.

C. Symptoms.

Swelling and redness, 2 or 3 days after birth, followed by a discharge which becomes creamy.

D. Complications.

Leucoma, anterior staphyloma or destruction of the eyeball.

E. Course.

Two to six weeks, followed by a chronic conjunctivitis.

F. Prognosis.

Good, if treated early, before the cornea is affected.

G. Prophylaxis (Crédé Method).

1 to 2% solution of silver nitrate is dropped into the conjunctival sac at birth. Salt solution is used immediately after. This procedure has greatly reduced the percentage of blindness since it has become obligatory.

VI. GONORRHEAL CONJUNCTIVITIS.

A. Synonyms.

Blennorrhoea; purulent conjunctivitis in the adult.

B. Definition.

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A serious, purulent conjunctivitis due to infection by the gonococcus.

C. Etiology.

The gonococcus of Neisser. May be conveyed to the eyes from discharges and infected articles.

D. Symptoms.

Swelling and tension of the eyelids. Purulent discharge oozing from between the lids. Conjunctiva thickened. Little pain but great discomfort. Cornea soon becomes infiltrated and ulcerated. The ulcers perforate, causing destruction of the eye, or they may cause corneal opacities.

E. Course.

One eye first affected. The other may not be if carefully shielded. Runs from two to six weeks, followed by a chronic conjunctivitis with a thick, granular conjunctiva.

F. Prognosis.

Grave.

G. Treatment.

Medical and prophylactic.

VII. CHRONIC FOLLICULAR CONJUNCTIVITIS.

A. Synonym.

Follicularis.

B. Definition.

A chronic affection of the palpebral conjunctiva, characterized by follicles, and very few, or no signs, of inflammation.

C. Etiology.

Occurs in the young, especially strumous (scrofula or tuberculosis of lymph glands) children, living under unhygienic conditions. It may also be

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infectious.

D. Pathology.
The follicles are lymphoid tissue masses, resembling trachoma follicles.

E. Subjective Symptoms.
Resemble those of a mild, chronic catarrhal conjunctivitis.

F. Objective Symptoms.
The follicles are found in the fornix and nasal region of the lower lid, and in the conjunctiva of the upper lid at the margin and ends of the tarsal plate. They appear as small, round, pale elevations.

G. Diagnosis.
May be mistaken for trachoma.

H. Treatment.
Medical.

VIII. TRACHOMA.

A. Synonyms.
Granular conjunctivitis; Granulated lids; Egyptian ophthalmia.

B. Definition.
A contagious disease of the conjunctiva, in which granulation is a prominent feature.

C. Etiology.
Believed to be a bacterium discovered by Richards in 1927 and isolated by the Rockefeller Institute. The disease is found among the poorer classes. It is more common among Jews and Irish. It is also common among the American Indians.

D. Pathology.

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Trachoma follicles have a scanty connective stroma, with lymph cells and incomplete capsule, and are imbedded in the conjunctiva.

E. Symptoms.

Photophobia, lacrimation, sticking of lids, mucous or muco-purulent discharge. When a lid, especially the upper, is everted, masses of irregular projecting granulations will be seen, gray in color and about the size of grains of sage.

F. Complications.

Pannus and corneal ulcers.

G. Sequelae.

Ectropion, entropion, trichiasis, symblepharon, corneal opacities, staphyloma, etc.

H. Course.

Chronic.

I. Treatment.

Prophylactic, medical and surgical.

J. Differential Diagnosis:

Chronic Follicular Conjunctivitis	Trachoma
1. Occur mostly in youth.	1. Any age.
2. Granulations small, round, and in rows.	2. Granulations larger, but less prominent.
3. Occur mostly at nasal side of lower lid and at edge and extremities of upper tarsal plate.	3. More evenly distributed.
4. Amenable to treatment; and granulations leave without a trace.	4. When treated and relieved, granulations leave scars.

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IX. PHLYCTENULAR CONJUNCTIVITIS.

A. Synonyms.

Scrofulous ophthalmia; Eczematous conjunctivitis.

B. Definition.

A disease of the bulbar conjunctiva, characterized by small papules or postules.

C. Etiology.

Probably due to a micro-organism often accompanied by nasal catarrh. The same condition is often seen on the face at the same time. Occurs in strumous children and in the ill-nourished.

D. Subjective symptoms.

Photophobia, irritation and lacrimation. If the phyctenules are not near the cornea, the child complains but little.

E. Objective symptoms.

One or more postules surrounded by an area of congestion appear on the bulbar conjunctiva. The postules may break down and form ulcers. The phyctenules may form in rapid succession, each lasting a week or so. Relapse is common. If the lesion occurs at the margin of the cornea, the condition is known clinically as phlyctenula marginalis.

F. Treatment.

Medical (general and local).

X. MEMBRANOUS CONJUNCTIVITIS.

A rare disease occurring in two forms:

A. Croupous Conjunctivitis.

It is the most common. There is a membranous deposit which leaves a bleeding surface if removed. It may be a complication of a severe conjunctivitis in children, of infectious diseases, or it may fol-

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low superficial burns.

B. Diphtheritic Conjunctivitis.
Caused by the Klebs-Loeffler bacillus. The lids are swollen, tender, red and stiff. The palpebral conjunctiva presents a dirty yellow diphtheritic membrane. The general symptoms of diphtheria are present. Necrosis occurs, which results in granulations and cicatrices, which deform the lid. The cornea and the whole eye may be affected.

XI. INJURIES OF THE CONJUNCTIVA.

They consist of foreign bodies on the conjunctiva; wounds; and burns.

XII. MISCELLANEOUS DISEASES OF THE CONJUNCTIVA.

A. Spring Catarrh.

1. Synonyms.
Conjunctiva aestiva; Vernal catarrh.

2. Definition.
A recurrent, uncommon disease, showing the presence of nodules around the edges of the cornea, together with hard, pale, flat granulations on the conjunctiva of the upper lid.

3. Etiology.
Unknown.

4. Symptoms.
Some irritation, photophobia and a sensation of foreign bodies. The disease appears in Spring, Fall or Summer; disappearing during the Winter.

5. Treatment.
Medical, but unsatisfactory.

B. Symblepharon.
A union of the conjunctiva of the lid and eye-

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ball producing adhesion. It follows wounds, burns and trachoma.

C. Pinguecula.

A small, yellow nodule on the bulbar conjunctiva, near the cornea, and as a rule on the nasal side. It is a hyaline degeneration and is common in middle and old age. Occasionally it becomes inflamed. No treatment necessary.

D. Pterygium. (ae)ter ig ium

1. Definition.

A membranous growth with its base near the inner or outer canthus and with its apex toward the center of the cornea.

2. Etiology.

It is formed of hypertrophied conjunctiva. Some cases are believed to be an extension of pinguecula.

3. Symptoms.

The patient complains only when the condition is advanced to the point of producing astigmia or of reducing vision. The growth occurs usually on the nasal side. If it is non-progressive it is dry, thin and non-vascular. The progressive type is thick and congested.

4. Course.

Many years.

5. Treatment.

Surgical.

E. Chemosis of the Conjunctiva.

Oedema of the ocular conjunctiva, accompanying violent inflammations of the eye.

F. Xerosis.

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Two different affections:

1. Advanced cicatricial contraction of the conjunctiva which is dry. It follows severe conjunctival diseases such as trachoma.

2. White plaques occur on the ocular conjunctiva probably caused by the Xerosis bacillus. The deposit is membranous and greasy appearing.

The first cannot be treated; but the second can be treated medically and surgically.

G. Ecchymosis of the Conjunctiva.

An extravasation of blood beneath the conjunctiva following rupture of the blood vessels. Caused by injury, and coughing; and sometimes appears spontaneously, in elderly people.

H. Pemphigus of the Conjunctiva.

Blisters occur and are followed by scar tissue which destroys the conjunctiva.

I. Tumors of the Conjunctiva.

Cysts, dermoid, angioma, papilloma, lipoma, fibroma, epithelioma and sarcoma.

= incapsulated fluid
= Tumor containing hair ~~scales~~ nails
= tumor composed of blood
= Endothelium tumor or cauliflower tumor
= embryonic

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Natures way of building new tissue.

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CHAPTER VIII.

DISEASES OF THE CORNEA.

I. ULCER OF THE CORNEA.

A. Definition.

A superficial loss of substance, with infiltration of the surrounding cornea.

B. Etiology.

General ill-health; or lowered nutrition of the cornea. Common among the poorer classes. May be produced by a foreign body or may be associated with diseases of the lacrimal apparatus and conjunctiva.

C. Pathology.

The cornea becomes infected and the superficial layers necrosed. The following organisms may be the infective agents, viz.: streptococcus, staphylococcus, pneumococcus, Morax-Axenfeld bacillus, aspergillus and gonococcus.

D. Varieties.

1. Simple or non-progressive.
2. Infected or progressive.

E. Clinical varieties.

1. Phlyctenular ulcer (in phlyctenular Keratitis)
2. Traumatic ulcer: from abrasions or wounds.
3. Serpent ulcer (Saemisch ulcer): infective ulcer with advancing crescent edge.
4. Dendritic ulcers: branched, superficial, infective ulcer.
5. Ring ulcer: encircling the periphery of the cornea.
6. Indolent or absorption ulcer: no signs of inflammation, occurs in marasmic infants, in the

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aged and those in ill-health.

7. Catarrhal ulcer: accompanies catarrhal conjunctivitis.

F. Subjective Symptoms. ——— = main Symp

Lacrimation, photophobia, sensation of foreign bodies, pain, and interference with vision if the ulcer is in the pupillary area.

G. Objective Symptoms.

Circumcorneal injection, and some conjunctival congestion. The ulcers vary in shape, appearance and extent. Oblique illumination will reveal a loss of substance and a grayish opacity.

H. Course.

SIMPLE ~~Single~~ ulcers heal in a week or so. Infected ulcers may spread and attack the healthy tissues, penetrating the lower layers and even perforating the cornea. When healing begins, vascularization of the cornea may occur.

I. Complications.

Opacities, adherent leucoma (iris is caught in the scar), staphyloma with prolapsed iris, hypopyon of the aqueous chamber, iritis, panophthalmitis, etc.

falls forward Pus

J. Treatment.

Medical: local and systemic.

II. INTERSTITIAL KERATITIS.

A. Synonym.

Diffused parenchymatous keratitis.

B. Definition.

A chronic inflammation of the cornea characterized by deep opacities and circumcorneal injection.

abnormal amt of blood present.

C. Etiology.

Commonly occurs in children having congenital

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syphilis. Rarely seen in acquired syphilis. Also may be due to gout, rheumatism, malaria, rachitis and tuberculosis and also may be idiopathic. rickets

D. Pathology.

Infiltration by leucocytes and sometimes deep vascularization at the margin of the cornea.

E. Subjective Symptoms.

Poor vision, photophobia and some pain. Discomfort

to bright light

F. Objective Symptoms.

Opacities, usually as a gray cloud at the margin. They may spread and cover the cornea, appearing white, mottled or yellow. Deep vascularization from the scleral vessels, causing a red spot (salmon patch). Surface is rough, dull and steamy. Rarely any ulceration, although the cornea may weaken and form a staphyloma.

G. Constitutional Objective Symptoms.

Of congenital syphilis; Hutchinson's teeth (notched margins); scars at angles of mouth and forehead; face prematurely wrinkled; head square and large; bridge of nose flat; chronic nose and ear diseases.

H. Course.

Both eyes affected as a rule. Disease occurs between five and fifteen years, although it may be delayed until the age of thirty years. Course is slow: (two months to a year). Often the opacities clear up but impaired vision occurs in several cases. Relapses often occur.

I. Complications.

Inflammations of the uveal tract. { choroid Sclera Retina

J. Treatment.

Medical: local and systemic.

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III. PHLYCTENULAR KERATITIS.

A. Definition.
A disease characterized by small pustules on the cornea.
——— = small yellow spots

B. Etiology.
Strumous children and the undernourished. Rarely seen in adults. May be of bacterial origin.

C. Varieties.

1. Vesicular Keratitis: an advancing curved infiltration with vascularization from it to the margin of the cornea. It may creep across the cornea leaving opacities.

2. Multiple ulcers with vascularization.

D. Symptoms.
Tonic blepharospasm. Photophobia so great that the patient completely covers his eyes.
~~~~~ = closes eyes.

E. Treatment.
Medical: local and systemic.

IV. STAPHYLOMA OF THE CORNEA.

A. Synonym.
Ectasia cornea.

B. Definition.
A bulging of the cornea, not due to hypertrophy or swelling of its tissues.

C. Etiology.
Follows weakening of the cornea by disease. May be partial or total, the latter preventing closure of the lids. The eye is usually blind from previous pathological conditions.

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~~~~~

D. Treatment.
Surgical.

V. KERATOCONUS.

A. Synonym.
Conical cornea.

Cause = maybe Malnutrition + tissue loses firmness

B. Definition.
A gradual bulging of the transparent cornea, assuming a conical form with the apex at or near the center. Usually begins in youth, but may occur at any age. There is no inflammation and it may become stationary.

C. Subjective symptom.
Increasingly defective vision.

D. Objective symptoms.
When marked, can be readily seen by a side view. When slight, a reflex from a window appears distorted, being lengthened on every side from the apex. There are high astigmia and myopia present. The apex may show an opacity; and ulcerate.

E. Treatment.
Non-progressive cases may show improved vision with cylinders. Progressive cases require surgical treatment.

VI. INJURIES, FOREIGN BODIES AND WOUNDS.

A. Abrasions.
The anterior epithelial layers are torn off, causing pain, photophobia and lacrimation.

B. Burns.
May be caused by chemicals (especially acids and alkalies), steam, hot metals and hot water. If the burns are superficial, they heal readily. If they are deep, the scars may affect vision.

Nerve endings of Cornea end in epithelium layer (2 layers down).

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C. Foreign bodies.

These range from particles of dust to pieces of steel. Use oblique illumination and magnifying glass.

D. Perforating wounds.

They may open the anterior chamber and allow the aqueous to escape. If it is a simple, non-infective puncture, healing occurs in a few days with a resulting scar. Infected cases may produce grave inflammatory conditions.

VII. MISCELLANEOUS DISEASES OF THE CORNEA.

A. Superficial Keratitis (Vascular Keratitis or Pannus).

Vascularization and infiltration of the cornea. Often complicates trachoma. Newly formed Blood Vessels

B. Herpes Zoster Ophthalmicus.

A herpes of the cornea when the 5th nerve is affected by herpes. Scars are left on the cornea.

~~~ = Multiple Eruptions Cornea Punctate Dotted

C. Herpes Corneae.

Recurrent eruption, of several hours duration, of small vesicles on the corneal surface. It causes pain, irritation and the sensation of foreign bodies. The symptoms disappear when the vesicle ruptures. It may follow corneal injuries and abrasions.

D. Keratitis Bullosa.

Large blisters occurring usually on the cornea of a diseased eye.

E. Sclerosing Keratitis.

A dense white opacity of the cornea, accompanying scleritis.

F. Filamentous Keratitis.

An ulceration with threads attached at one end.

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~~~

G. Keratitis Profunda.

A deep central interstitial inflammation in the adult. It may be due to malaria, rheumatism or exposure. Itis in stroma layer.

H. Ribbon-shaped Keratitis (or transverse calcareous film.)

A grayish-white band horizontally crossing the cornea. It is hard and contains lime. Follows eye-diseases or degeneration.

I. Pigmentation of the Cornea.

Stain from blood pigment or from the presence of iron or steel (piderosis).

J. Neuroparalytic Keratitis. → Changes

Caused by a lesion of the 5th nerve, characterized by ulceration, necrosis and anesthesia, which are caused by undetected foreign bodies and trophic changes.

K. Posterior Punctate Keratitis (Descemetitis).

Minute triangular shaped deposits, with the base down, occurring on the lower portion of the posterior surface of the cornea. It is a manifestation of uveal diseases. → Mid or all coats

L. Superficial Punctate Keratitis. → Most general.

There are small elevated opacities on the anterior part of the cornea with irritation and congestion of the eye.

VIII. TUMORS OF THE CORNEA.

These are rare.

Sarcoma, epithelioma, dermoid, fibroma and papilloma have been described, occurring mostly at the limbus. ↓ Start eg.

IX. ARCUS SENILIS (GERONTOXON).

A ring-like opacity at the margin but with a narrow zone of clear cornea between it and the sclera.

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It is a fatty degeneration or hyaline deposit occurring in elderly people. It is not pathological as a rule.

X. ARCUS JUVENILIS. Lymph tends to coagulate.

Occurs in the young and it may indicate vascular disease.

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CHAPTER IX.

DISEASES OF THE SCLERA.

I. SCLERITIS.

A. Definition.

A local inflammation characterized by elevated, congested and discolored patches in the sclera.

B. Etiology.

Occurs in adults, especially those subject to rheumatism, malaria, gout, syphilis or tuberculosis, or it may be idiopathic.

C. Varieties.

1. Episcleritis: involving the superficial sclera.
2. Scleritis: involving the whole depth of the sclera.

D. Symptoms of Episcleritis.

Moderate photophobia, irritation and pain. A bright red or violet patch is seen on the sclera near the cornea. Frequent relapses.

E. Symptoms of Scleritis.

Serious form: quite severe pain, tenderness, lacrimation.

Red, yellow or violet, elevated patches on the sclera, which may encircle the cornea.

The cornea, iris and ciliary body may be involved.

F. Complications.

Keratitis, cyclitis, iritis and anterior uveitis.

The disease may thin and weaken the sclera, producing staphyloma from internal pressure.

Glaucoma may occur.

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Scleritis may leave permanent dark-bluish spots at the site of the lesion.

G. Treatment.

Medical: local and systemic.

II. STAPHYLOMA.

A. Definition.

A bulging of the sclera without any thickening of its tissues.

B. Causes and Symptoms.

It follows scleritis.

Injury that weakens the cornea and sclera.

Occurs in diseases of the ciliary body and choroid when increased tension is present.

It occurs as dark-bluish elevations.

C. Treatment.

Surgical.

III. POSTERIOR STAPHYLOMA.

A. Definition.

Bulging of the sclera around the optic nerve.

It is associated with high myopia. Not Pathological

B. Treatment.

Surgical.

IV. INJURIES OF THE SCLERA.

Injuries may produce rupture of the eyeball.

Incised wounds, if large, may be accompanied by prolapse of the ciliary body and choroid, or by loss of the vitreous.

Infection may follow which may result in panophthalmitis or shrinking of the eyeball. (Phthisis bulbi).

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CHAPTER X.

DISEASES OF THE IRIS.

I. GENERAL STATEMENTS.

A. Varieties.

1. Acute. – one which appears quick & leaves same.
2. Subacute. – " at which has been acute, then pains.
3. Chronic. – long drawn out disease & may continue throughout life.

B. Causes.

1. Syphilitic.
2. Gonorrheal.
3. Rheumatic. = a common cause in elderly people.
4. Tuberculosis.
5. Traumatic. = Injury to eye.
6. Secondary.
7. Idiopathic. = true facts are not known.
8. Sympathetic. = one eye has been affected & then to other

C. Pathology.

1. Serous. = in forms of flakes
2. Purulent. = pus
3. Plastic. = increase in amt of tissue.

II. ACUTE PLASTIC IRITIS.

A. Definition.

An iritic inflammation, characterized by a small pupil, congestion and posterior synechia.

B. Etiology.

Syphilis and rheumatism are the most frequent causes.

Injuries, gout, diabetes, keratitis, scleritis and idiopathic.

adhesions of Iris to Cut surface of ~~Cornea~~ Lens

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C. Pathology.

Iris swollen and congested.

Exudate collects in the anterior chamber and causes adhesion of the pupillary margin and the posterior surface of iris to the lens capsule.

D. Subjective Symptoms.

Severe pain in temple, eye and forehead, which is worse at night.

Photophobia and lacrimation.

Vision is affected.

Constitutional symptoms may be present.

E. Objective Symptoms.

Lids red and swollen.

Circumcorneal injection.

Cornea hazy and sometimes covered with minute dots on its posterior surface.

Anterior chamber cloudy, with deposits on lower half of the iris, sometimes.

Anterior surface of iris is muddy and dirty colored (compared with the other eye).

Pupil small and hardly reacts to light.

Dilation is irregular (posterior synechia).

Fundus obscured.

F. Course.

Begins as an acute condition, lasting one to six weeks.

Uncommon in children, but may occur at any age.

It tends to recur.

G. Prognosis.

Early treatment admits of excellent prognosis.

H. Complications.

Occlusion of pupil by synechia.

Crater-shaped pupil.

Glaucoma, cataract, iridocyclitis, irido-choroiditis and anterior uveitis.

In syphilitic iritis, yellow nodules may occur

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at the periphery or the pupillary margin.

I. Diagnosis.

By the pain, which is worse at night; muddy iris; posterior synechia; small pupil and ciliary congestion. → Adhesion of Post to Ant.

J. Differential diagnosis.

See table under glaucoma.

K. Treatment.

Darkened rooms: medical treatment; general and local.

III. CHRONIC PLASTIC IRITIS.

It commonly occurs in elderly rheumatic patients. The attacks are not severe, but occur often. Each attack thickens the iris, especially the pupillary margins, causing occlusion, glaucoma and destruction of the eye.

Treatment is medical.

IV. SEROUS IRITIS.

A. Synonyms.

Keratitis punctata posterior; Descemitis; Aquocapsulitis.

B. Definition.

A serous inflammation, not only of the iris, but including the ciliary body, sometimes the choroid, and the endothelial layer of the cornea. → one which has lot of exudate.

C. Symptoms.

Slight ciliary congestion.

Deposits of various sizes on the posterior surface of the cornea. These deposits are grouped in triangular manner, base down, in the lower half of the cornea.

The anterior chamber is deep and the pupil enlarged. → iris appears far back.

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D. Treatment.

Medical: local and general, as for Plastic Iritis.

V. PURULENT IRITIS.

Definition:

A purulent inflammation of the iris, usually caused by perforation; although it may be metastatic.

Pus → Hypopyon is present. → Spread of infection

It may occur as a complication of purulent conditions of the vitreous and choroid.

VI. TUBERCULAR IRITIS.

A. Forms:

1. Isolated tubercles.

Yellow tumors at the outer margin of the iris.

Increase in size and involve other structures destroying the eye.

2. Miliary tubercles.

There is an acute iritis, but with little pain.

There are minute elevations covering the surface of the iris.

It eventually destroys the eye.

3. Tuberculosis of the eye is rare.

B. Treatment.

Medical, local and general.

Surgical (enucleation).

VII. INJURIES OF THE IRIS.

A. Blows upon the eyeball may cause a regular or irregular dilatation due to paralysis of the sphincter pupillae.

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B. Perforating wounds: usually accompany injuries of other structures of the eye.

VIII. TUMORS OF THE IRIS.

Most Common

Rare: they include sarcoma, melanoma and cysts.

Pigmented tumor Black Spots.

IX. CONGENITAL DEFECTS OF THE IRIS.

A. Irideremia or Aniridia: congenital lack of the iris.

B. Coloboma of the iris: congenital absence of a part of the iris, usually at the inferior nasal side.

C. Corectopia: irregularly placed pupil.

D. Policoria: multiple pupil. Eg:→

E. Persistent pupillary membrane: Remnants of the foetal pupillary membrane.

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CHAPTER XI.

DISEASES OF THE PUPIL.

I. PARALYTIC MYDRIASIS. = dilatation

Due to a lesion of the third nerve or its nucleus (locomotor ataxia, disseminated sclerosis, hemorrhage, tumors and injuries), of the optic nerve or tract, and paralysis of the sphincter pupillae.

II. SPASMODIC MYDRIASIS.

Due to high intracranial pressure, mental excitation or spinal irritation.

III. PARALYTIC MYOSIS.

Due to diseases of the superior part of the spinal cord, and tumors or wounds of the cervical sympathetic.

IV. SPASMODIC MYOSIS.

Meningitis; irritation or lesion of the third nerve or its nucleus. Foreign body irritation.

V. ARGYLL-ROBERTSON PUPIL.

Occurs mostly in locomotor ataxia.

Pupil contracts to accommodation but not to light.

VI. HIPPUS.

Alternate contraction and abnormal dilatation.

Depends on nervous condition.

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CHAPTER XII.

DISEASES OF THE CILIARY BODY.

Rarely affected alone: the iris or choroid or both may be involved.

I. DIFFERENCES BETWEEN CONJUNCTIVAL AND CILIARY INJECTION.

A. CONJUNCTIVAL INJECTION	CILIARY INJECTION
1. Diseases of the conjunctiva.	1. Diseases of iris, ciliary body, cornea.
2. Mucous or muco-purulent discharge.	2. Lacrimation. No discharge.
3. Most marked in fornix.	3. More marked around cornea.
4. Fades as it approaches cornea.	4. Fades as it approaches fornix.
5. Bright red in color.	5. Pink or lilac in color.
6. Network of vessels.	6. Vessels appear straight.
7. Vessels can be moved with the conjunctiva.	7. Vessels cannot be moved with the conjunctiva.

B. Cyclitis.

Varieties:

1. Plastic.
2. Purulent.
3. Serous.

important

II. ACUTE PLASTIC CYCLITIS.

A. Definition.

Acute inflammation of the ciliary body with exudation.

B. Symptoms.

Pain and tenderness in ciliary region.
Circumcorneal congestion.
Some opacities in the vitreous.
Glaucoma may be a complication.

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If the iris is involved, there are symptoms of iritis.

If choroiditis is present, patches of exudate will be seen, if the vitreous is sufficiently clear.

C. Causes.

Same as iritis.

D. Prognosis.

Bad if disease is severe.

Permanent occlusion of pupil and opacities in the vitreous may result.

III. CHRONIC PLASTIC CYCLITIS.

Definition and Symptoms:

Iris, as a rule, is involved.

The pupil is occluded and there is exudate in the vitreous.

The exudate back of the lens tends to organize, and draw the ciliary body together, and the outer margin of the iris is retracted.

Traumatic, chronic, plastic cyclitis may cause sympathetic ophthalmia.

IV. SEROUS CYCLITIS.

This is the same as serous iritis, as both iris and ciliary body are involved.

V. PURULENT CYCLITIS.

A. Definition.

A purulent inflammation involving all of the uveal tract. Usually follows perforating wounds.

May occur by metastasis from meningitis or diseases of the nose.

B. Treatment.

Medical: local and systemic.

VI. TUMORS OF THE CILIARY BODY.

Rare: sarcoma, tubercles, gummata and cysts.

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Table of Cataracts

- General Subdivision
  - Primary
  - Secondary
- according to lens involved
  - Lenticular
  - capsular
  - Capsulo-Lenticular
  - Partial
  - Complete
  - Stationary
    - Ant. polar or pyramidal
    - Post. Polar
    - Zonular
    - Fusiform, Central, Punctate
  - Progressive
    - Senile
    - congenital
    - Traumatic
- according to Etiology
  - congenital
    - Ant. Polar
    - Post. Polar
    - Lamellar or Zonular
    - Occational Complete cataract
  - Senile (simple) (age)
  - Diabetes (Rumon)
  - Nephritis
  - Gout
  - General Arterial Diseases
  - } less frequent
  - Traumatic
  - Due to Ocular Diseases
    - Irido Cyclitis
    - Ulceration Keratitis
    - Choroiditis
    - Hi Myopia
    - Detachment of Ret.
- accord to age
  - Hard
  - Soft

CHAPTER XIII.

DISEASES OF THE LENS.

I. CATARACT.

A. Definition.

An opacity of the crystalline lens or its capsule.

B. Varieties.

Senile, traumatic, zonular, polar, posterior and anterior (including capsular).

Progressive (senile and traumatic) and stationary (polar and zonular).

Primary (without any known disease of the eye) and secondary (when associated with some disease of the eye).

II. ANTERIOR POLAR CATARACT.

A. Synonym.

Pyramidal cataract.

B. Definition.

An opacity at the anterior pole of the lens.

C. Subjective symptoms.

Usually little interference with vision.

D. Objective symptoms.

Oblique illumination reveals a small round dense opacity at the anterior pole, often elevated. It extends into the substance of the lens.

An opacity of the cornea often is seen near the center.

E. Etiology.

Congenital or acquired.

It is often due to contact of the lens with the cornea, following perforation of an ulcer either

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before or just after birth.

F. Treatment.
None.

III. POSTERIOR POLAR CATARACT. ONE AT POST. SURFACE of LENS. or POLE

A. Definition and symptoms.
Similar to anterior Polar cataract.
Occurs in congenital and acquired forms.

1. Congenital form.
Small round opacity, due to contact of the hyloid artery with the lens. It may sometimes be found as a minute dot in normal eyes.

2. Acquired form.
Involved in intra-ocular diseases; often of the choroid.

IV. LAMELLAR ZONULAR CATARACT.

A. Definition.
An opacity, consisting of one or more zones, which surround a clear nucleus and leave the outside clear.

B. Symptoms.
By oblique illumination, streaks are seen running out into the clear cortex.
There is considerable variation in the extent of this type of cataract.
Congenital form may show complete opacity.
A stellate or punctiform type about the nucleus, may occur.

C. Course.
Nearly always remains stationary.

D. Etiology.
Usually congenital, occurring in children who

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State Board Question:

What are the (3) Pathological conditions according to Fremwall?

(1) The lens fibres die very slowly & become sclerosed. Sclerosis is uneven & interfers with vision.

(2) The lens fibres die very rapidly & become autolyzed (dissolving). In the resulting fluid a precipitate is formed which may be either amorphous or crystalline in character. The precipitate is the basis of the opacity.

(3) Due to some unknown irritation. The cells of the anterior chamber or capsule proliferate & form an opacity of fibrous membrane which in some cases extends all around the lens.

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were rachitic or had convulsions in infancy. There is an apparent hereditary tendency.

E. Treatment.
Surgical.

V. SENILE CATARACT.

A. Etiology.
Most cataracts of this type occur in the aged. There appear to be no causes, although constitutional diseases favor their development.

B. Pathology.
Irregular shrinking of fibres, and collection of fluid in the spaces thus formed, during nucleus formation. The fibres degenerate and the fluid coagulates causing opacities.

C. Subjective symptoms.
Dark spots, streaks and flashes of light, blurred vision, diplopia.
No pain.
Eye-strain.
Sometimes first indication, is ability to see without glasses, due to the swelling of the lens increasing its refracting power.

D. Objective symptoms.
Stages:

1. Incipient Cataract.
Opacity at the center of the lens (nuclear) or radiating streaks (cortical).
Seen best by oblique illumination, where they appear white, or by the ophthalmoscope, when they appear black against a red background.

2. Immature Cataract.
Ripening process.
Opacity becomes more extensive and there is

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an increased swelling of the lens.
Opacity can be seen in day-light.

3. Mature Cataract. ✓
Ripe: fully opaque.
Returns to normal size.

4. Hypermature Cataract.
Cortex softens, and may become fluid, with the nucleus floating in it (Morgagnian Cataract), or the lens may become flat and may calcify.

5. Course.
Progress slow. ✓
May be years before maturity is reached, or it may become stationary.
Both eyes may or may not be affected at once.

6. Prognosis.
An eye free from disease, with the anterior chamber of normal depth, and a freely reacting pupil; a normal tension and projection good, when the cataract is ripe, will admit of a good prognosis for good vision after operation.

7. Treatment.
Surgical.

II. DISLOCATION OF THE LENS. By rupture of suspensory lig.

A. Definition.
There may occur partial (subluxation) or complete (luxation) dislocation of the lens by rupture of the suspensory ligaments.

B. Etiology and symptoms.
Traumatic, congenital and secondary.

1. Dislocation occurs upward, downward, backward or sideways into the vitreous chamber.

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With the ophthalmoscope, one may see a curved, black line in the pupil.
Eye movements shake the lens and cause tremulous iris.

2. The lens may be dislocated partly through the pupil or entirely into the anterior chamber.
Difficult to see.
Usually followed by glaucoma.

3. The lens may be dislocated through a wound in the sclera and lie under the conjunctiva.

4. A dislocated lens is liable to cause glaucoma.

C. Treatment.
Cataract operation, if inflammation or glaucoma is threatened, or present.

III. CONGENITAL LENS AFFECTIONS.

A. Coloboma.
Rare: portion of the lens is absent.

B. Lenticonus Posterior.
Rare: bulging of the posterior surface.

IV. APHAKIA.

A. Definition.
Absence of the crystalline lens.

B. Causes.

1. Congenital.
2. Result of cataract extraction.
3. Result of sub-luxation.

C. Mechanical Treatment.
Distance vision is possible only with the aid of a convex lens. If the eye were emmetropic before

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the operation, the hyperopia afterward would amount to an average of from 10D to 12D. If hyperopia was present previously, it is added to that which is acquired by the operation, and makes it proportionately greater. If, on the other hand, the eye is myopic before the operation, the hyperopia after cataract operation, is that much less. Extremely myopic eyes may actually become emmetropic or even remain myopic.

The aphakic eye is destitute of accommodation, hence, it follows that by single glasses, the latter is corrected for a single distance only. The eye needs at least two pairs of glasses; one for distance and the other for close work.

Owing to the alteration in the corneal curvature, produced by the contraction of the operation scar, usually a considerable amount of astigmia (from 1D to 4D) against the rule, is produced by the cataract operation. This usually diminishes during the first few months so that the final correction by glasses may be materially different from the first correction. Since the adoption of the scleral incision, this astigmatism is not so marked.

[Note: As a good practical rule, glasses should not be prescribed for a month or more after the crystalline lens has been removed.]

Katral lens used. 8D. to 20 D.

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CHAPTER XIV.

DISEASES OF THE VITREOUS.

I. OPACITIES.

A. Fixed Opacities.

Remains of the hyaloid artery in its canal.

Connective tissue bands, either congenital, or resulting from organization of inflammatory exudate.

B. Floating Opacities.

Dust-like (syphilis).

Masses, bands etc. from cyclitis, choroiditis, retinitis, hemorrhages, injury and degeneration of the vitreous.

The degenerative changes in the vitreous occur in high myopia, constitutional weakening diseases, old age, systemic disease, menstrual disorders; or they may be idiopathic.

C. Muscae Volitantes.

Subjective floating opacities.

They are shadows of vitreous cells thrown on the retina.

D. Sinchysis scintillans.

Cholestrin crystals, or scales in the vitreous, which reflect the light as brilliant, floating spots.

II. SUPPURATIVE INFLAMMATION OF THE VITREOUS.

A. Synonym.

Purulent hyalitis.

B. Etiology.

Pus in the vitreous due to infection from wounds, or by metastases from meningitis, ear and nose diseases, or general infectious diseases, in-

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flammations of the uveal tract and from debilitating diseases.

C. Symptoms.

Lens, aqueous and cornea are clear.
There is a yellow reflex back of the lens.
In advanced cases the eye is soft.
Severe cases are known as panophthalmitis or abscess of the eye.

D. Treatment.

Surgical.

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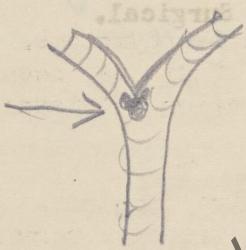
Thrombus = plugging of vein by clot & occurs after death.

Embolism: eg:



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CHAPTER XV.

DISEASES OF THE RETINA .

I. ANEMIA OF THE RETINA.

It may be an ocular symptom of a general condition, or it may be local.

A. Etiology.

Embolism, compression, cardiac and vascular diseases and poisons.

Plugging of Vessel, by clot. (ART.)

Pressure on brain by lymph.

B. Subjective Symptoms.

There may occur some decrease in vision, with contraction of visual fields, headache and blindness.

C. Objective Symptoms.

Arteries very narrow, disc extremely pale.

A chronic form occurs after retinal disease, causing atrophy.

The vessels are very narrow and bordered by white lines of connective tissue or the vessels may appear empty and threadlike.

D. Treatment.

Medical.

II. HYPEREMIA OF THE RETINA.

A. Causes.

Asthenopia, excessive light and heat.

B. Objective Symptoms.

When slight, the disc is slightly redder than normal, with a slight striation of its margins.

When marked, there may be present, retinal or other ocular inflammation.

C. Treatment.

Medical and mechanical.

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III. SIMPLE RETINITIS.

A. Synonym.
Retinitis in general.

B. Definition.
Inflammation of the retina.

C. Etiology.
Sometimes obscure.
Commonly occurs in constitutional diseases, such as malaria, leukemia, anemia, arterio-sclerosis, etc. White disease

D. Subjective Symptoms.
Impaired vision, blurring, flashes of light.
Sometimes photophobia and metamorphopsia are present. distorsion of Ret.

E. Objective Symptoms.
Retina may be only slightly affected, with dilated veins, tortuous vessels and few hemorrhages.
Severe cases show a cloudy fundus, dilated and distorted vessels deep in the swollen retina, flame-like hemorrhages and blurring of the outline of the disc.
The disease may persist for months and may occur in one or both eyes.

F. Treatment.
Medical.

IV. HEMORRHAGIC RETINITIS.
This disease is a form of simple retinitis with marked hemorrhages. Thrombosis of the retinal veins, or hemorrhages between the retina and the choroid, may occur.
The disease is most common in elderly people suffering from arterio-sclerosis.

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V. ALBUMINURIC RETINITIS.

A. Definition.

A retinitis occurring with acute or chronic nephritis.

Both eyes are usually affected.

B. Etiology.

Nephritis and Bright's disease of pregnancy.

C. Subjective Symptoms.

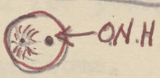
Interference with vision often very slight.

Nephritic patients are subject to attacks of uroemic, temporary blindness with or without retinitis.

D. Objective Symptoms.

There are signs of simple retinitis, with shining white patches throughout the fundus.

There is a peculiar stellate figure formed by radiating lines of glistening white dots around the macula.



E. Pathology.

The white patches are areas of fatty degenerations of retinal elements, and exudates.

F. Prognosis.

When due to chronic nephritis, it is a late manifestation, and the patient rarely lives two years after the eye lesion has appeared.

G. Treatment.

Medical.

VI. SYPHILITIC RETINITIS.

A. Etiology.

Congenital syphilis and acquired syphilis in the second and third stages.

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B. Subjective Symptoms.
Same as for other types of retinitis.

C. Objective Symptoms.
Dust-like opacities in the retina.
There is a bluish-gray haze over the retina, around the disc and macula.
Streaks of white exudate along the vessels.

D. Course.
Chronic.
Leads to atrophy of the optic nerve and choroiditis.

E. Treatment.
Medical.

VII. RETINITIS PIGMENTOSA.

A. Definition.
A disease of the retina beginning in youth and prolonged for years.

B. Subjective Symptoms.
Nyctalopia (night-blindness or loss of vision in subdued light)
Field of vision gradually narrows.

C. Objective Symptoms.
Masses of retinal pigment, irregular in shape, but with branching projections.
First appear at the periphery, later approach the disc.
The nerve and retina become atrophic.
Vessels reduced in caliber.

D. Course.
Years.
Hereditary tendency.
Consanguinity of parents involved.

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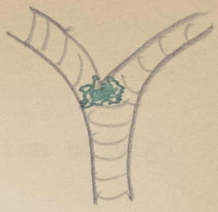

VIII. EMBOLISM OF THE CENTRAL RETINAL ARTERY.

A. Definition.

Plugging of the artery, or more rarely, a single branch.

B. Etiology.

Heart lesions.
Obliterating endarteritis of retinal vessels.

C. Subjective Symptoms.

Sudden monocular blindness.
No pain or other symptoms.

D. Objective Symptoms.

Retina soon becomes foggy (oedema)
Cherry-red spot at the macula.
Arteries small.
Little blood in the veins.
If circulation is restored, blood appears in broken columns.
Sometimes central vision remains.
Atrophy of the nerve and retina is the usual result.

E. Treatment.

Surgical (Massage).

IX. DETACHMENT OF THE RETINA.

A. Synonym.

Ablatio Retinae.

B. Definition.

Separation of the retina from the choroid, leaving the retinal pigment attached to the choroid.

C. Etiology.

Extravasation of blood or serum.
Exudate or new-growth.
Changes in the vitreous.

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Ordinary forms are a complication of high myopia.

Traumatism is a frequent cause.

D. Subjective Symptoms.

Poor vision and defect in visual field corresponding to the detachment.

E. Objective Symptoms.

Opacities may be floating in the vitreous.

Retina appears as a wavy, grayish, or greenish-white membrane over which the dark-red retinal vessels run, disappearing abruptly to reappear again. They run a tortuous course.

Retina floats about with movements of the eye; and may be torn.

Tension of the eye reduced.

Cases are recognized showing flat detachment (hard to see) and steep (most common) detachment.

F. Prognosis.

If complicating myopia, it gets worse, until vision is lost.

If it follows injury, it may recover, or remain stationary.

G. Treatment.

Medical and rest.

X. GLIOMA OF THE RETINA.

A. Definition.

A malignant tumor occurring in early childhood, usually before five years of age.

B. Pathology.

It springs from the molecular layers of the retina. It consists of blood-vessels, small round cells, cells with processes and a small amount of stroma.

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C. Subjective Symptoms.

The eye is blind.

D. Objective Symptoms.

1st Stage.

Shining white or yellowish reflex ("amaurotic cat's eye"). A few blood vessels are seen on the mass.

2nd Stage. (Glaucomatous).

Eyeball becomes hard because of the increasing size of the tumor filling the eyeball.

3rd Stage.

New growth bursts through the eyeball either forward or backward.

4th Stage.

Metastatic growths in other organs. The other eye may become affected. The child dies of exhaustion or cerebral complications. Other children in the same family may be affected.

E. Differential Diagnosis.

Glioma	Pseudoglioma or Purulent Choroiditis
Occurs in early childhood.	Occurs at any age.
No history of injury or meningitis.	Follows injury or meningitis.
Tumor often well defined, with rest of vitreous clear.	Vitreous completely filled with yellowish mass.
Anterior chamber shallow.	Anterior chamber shallow.
Tention increased.	Tension decreased.
	Iris bulges at pupillary margin.
No inflammatory signs.	Early inflammatory signs.

F. Treatment.

Surgical. Early and complete enucleation of the orbit.

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XI. OPAQUE NERVE FIBERS.

Definition.

A congenital condition, in which may be seen brush-like, glistening, white patches around the optic nerve. They are composed of nerve fibers that have not lost their medullary sheaths. It is not pathological.

It is a normal condition of the rabbit's eye.

XII. INJURIES OF THE RETINA.

Commotio Retinae (Oedema).

Arises from contusions. There are defective vision and gray infiltration, especially in the macular region.

XIII. OTHER FORMS OF RETINAL DISEASE.

A. Snow Blindness.

From exposure to brilliant light. May cause retinitis, pigment changes, central scotoma and macular changes.

B. Retinitis Circinata.

White streak of exudate encircling the macula.

C. Retinitis Proliferans.

Masses of organized connective tissue in the vitreous. Probably due to hemorrhages.

D. Angoid Streaks.

Black or brown streaks in the deeper layers. Probably due to hemorrhages.

E. Retinitis Striata.

White streaks of fibrous tissue in the retina.

F. Amaurotic Family Idiocy.

In infancy there are changes in the macula. Hazy appearance in macular region with a red spot in the center. Probably due to degeneration of the

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ganglion cells, dependent upon the cessation of the development of the nervous system. Jews more susceptible to the disease. Children die in a year or two.

XIV. AMBLYOPIA.

A. Definition.

Functional blindness of the retina. There are no discoverable refractive errors or lesions.

B. Forms.

1. Congenital.

Associated with errors of refraction, notably hyperopia and astigmia. A squinting eye may be amblyopic, probably from non-use (amblyopia ex anopsia).

2. Hysterical.

One eye only affected as a rule. Partial or total. Field of vision contracted. Color fields reversed as to size.

3. Simulated.

Malingering.

4. Toxic.

Occurs in urcemia, malaria, drug, tobacco and alcohol poisonings. Lesions, particularly of the nerve may develop.

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CHAPTER XVI.

DISEASES OF THE CHOROID.

Varieties.

Of inflammation; exudative, serous and suppurative choroiditis and sclerochoroiditis posterior.

I. EXUDATIVE CHOROIDITIS.

A. Definition.

An inflammation of the choroid characterized by patches of plastic inflammation, followed by atrophy.

B. Pathology.

Exudate consists of round cells in the choroid and external retinal layers. It becomes organized, producing atrophy and pigment disturbances.

C. Etiology.

Syphilis most common, nutritional disorders; and rarely, tuberculosis. It may be idiopathic.

D. Varieties.

1. Central.

Occurs in syphilis, senility and myopia.

2. Disseminated.

Chronic. Scattered patches.

3. Diffuse.

Due to syphilis. Retina involved.

4. Isolated.

Not due to syphilis. Runs a short course. Result of over-exertion or may be idiopathic. Isolated patches.

E. Subjective Symptoms.

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Gradual loss of vision, although in some cases, sight remains good. Visual field contracted. Scotomata sometimes present.

F. Objective Symptoms.

1. Recent cases.
Show irregular, hazy, white or yellow patches. Isolated hemorrhages may occur.

2. Atrophic stage.
Masses of pigment, or white patches, which may or may not be ringed with pigment. Optic nerve atrophy, or opacities of the lens or vitreous, may be complications.

G. Treatment.
Medical.

II. SEROUS CHOROIDITIS.
A complication of Serous Iritis (q.v.)

III. SUPPURATIVE CHOROIDITIS.

A. Definition.
Practically the same as suppurative inflammation of the vitreous (q.v.). All structures of the eye may become involved and the eye destroyed.

B. Treatment.
Surgical.

IV. SCLEROCHOROIDITIS POSTERIOR.
A slow process of atrophy of the choroid around the optic nerve, usually toward the macula. A complication of myopia. Associated with posterior scleral staphyloma.

V. TUMORS OF THE CHOROID.
Secondary carcinoma, gumma, tubercles and sarcoma, (rare).

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VI. SARCOMA OF THE CHOROID.

A. Pathology.
Most common is melanosarcoma.

B. Symptoms.
1st Stage.
Blurred vision. Tumor projects into fundus carrying retina with it. Vessels can be traced over its surface without a break. Other than retinal vessels seen.

2nd Stage.
Eyeball is hard and painful and sight is lost.

3rd Stage.
Neighboring parts involved. It breaks through the eyeball or extends back through the optic nerve.

4th Stage.
Metastatic growths.

C. Diagnosis.
A rounded tumor, springing from the choroid, carrying the retina with it. The retinal vessels are unbroken over the surface of it, and there is increased tension.

D. Prognosis.
Grave.

E. Treatment.
Enucleation.

VII. INJURIES OF THE CHOROID.
These include perforation by wounds, and ruptures by contusion. The latter show a curved white line of the sclera. They are bordered by pigment, and usually they are around the optic nerve. No treatment possible.

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VIII. CONGENITAL DEFECTS OF THE CHOROID.

Coloboma.

Failure of the embryonic choroidal fissure to close. There is an exposed area of the sclera from the optic nerve toward the ciliary body.

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CHAPTER XVII.

DISEASES OF THE OPTIC NERVE.

I. OPTIC NEURITIS.

A. Synonym.
Choked disc, Papillitis.

B. Definition.
Inflammation of the optic nerve-head, characterized by congestion and swelling of the disc.

C. Etiology.
Brain tumors, nephritis, syphilis, anemia, rheumatism, vascular diseases, poisons, infectious diseases, orbital diseases, and sinus diseases.

D. Pathology.
White cell infiltration. It is believed that a distention of the optic nerve sheath is present.

E. Subjective Symptoms.
Defective vision. A peculiarity is, that severe cases may still have good vision.

F. Objective Symptoms.
Disc is congested or white in color. Edges streaked and blurred. Swelling of the disc. Veins distended and tortuous. Arteries small. May be hemorrhages. Field of vision may be defective. There may be a general retinitis. Both eyes are usually involved. In "choked disc," so called, there is great oedema of the nerve, dilated vessels and hemorrhages.

G. Course.
Months. It may clear up with no after-effects, or it may be followed by atrophy.

H. Treatment.
Medical.

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II. ACUTE RETROBULBAR NEURITIS.

A. Definition.

Inflammation of the orbital portion of the optic nerve.

B. Etiology.

Infectious diseases, rheumatism, poisonings (methyl alcohol), syphilis and following inflammation of neighboring structures.

C. Pathology.

The fibers supplying the macula only, are involved in most cases.

D. Symptoms.

Rapid loss of sight. Tenderness and orbital pain. There may be no ophthalmoscopic signs or only moderate optic neuritis. Optic atrophy is likely to follow, especially at temporal side of disc, with central scotoma.

E. Treatment.

Medical.

III. CHRONIC RETROBULBAR NEURITIS.

A. Synonym.

Toxic amblyopia.

B. Definition.

A condition in which there is a gradual loss of vision and atrophy of the temporal side of the disc.

C. Etiology.

Tobacco, especially in conjunction with alcohol, is the most common cause. Carbon bisulphide, lead, arsenic and other poisons also cause it. A disease of middle and late life.

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D. Pathology.

It is a chronic interstitial inflammation of the fibers supplying the macula, on the temporal side of the disc.

E. Symptoms.

Gradually failing vision. Diminished central vision and defect in the color perception around the point of fixation. Pallor of the temporal side of the disc and dilatation of retinal veins. Both eyes are affected.

F. Course.

Long. Does not produce total blindness.

G. Treatment.

Medical.

IV. ATROPHY OF THE OPTIC NERVE.

A. Definition.

Degeneration and shrinking of the optic nerve-fibers causing a white or gray disc.

B. Etiology.

1. Primary.

Idiopathic or accompanying brain and spinal cord diseases. May be hereditary. Begins in youth and gradually causes blindness.

2. Secondary.

Following optic neuritis, injuries, glaucoma, and diseases of the retina and choroid.

C. Pathology.

Chronic interstitial inflammation and atrophy of nerve-fibers.

D. Symptoms.

Gradual loss of sight and sometimes contrac-

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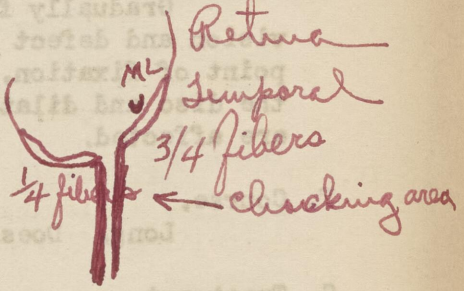
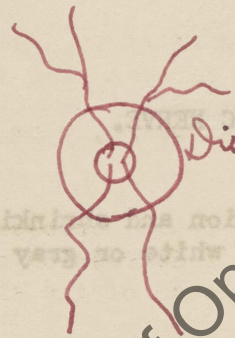
Ophthalmoscopy





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tion of visual field and defects in color vision. Disc may be gray, white or slightly pale. It shows a central depression with sloping sides in advanced cases.

E. Course.
Long.

F. Treatment.
Medical.

IV. TUMORS OF THE OPTIC NERVE.
The following are known to occur:- glioma; endothelioma, fibroma, myxoma, sarcoma, and tubercles.

V. CONGENITAL AFFECTIONS.

A. Inferior Conus.
A white crescent, usually on lower side of nerve.

B. Coloboma of the Optic Nerve-sheath.
There is a depression on lower side of disc. It is due to absence of the sheath.

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CHAPTER XVIII.

DISEASES OF THE ORBIT.

I. PERIOSTITIS.

A. Definition.

Inflammation of the membrane covering the orbital surfaces of the bones of the orbit.

B. Etiology.

Injuries, rheumatism, syphilis, tuberculosis, and extension from neighboring sinuses.

C. Symptoms.

Tenderness, pain and swelling. Abscess with fistula and contraction of tissue may occur. This is followed by cicatricial ectropion.

D. Treatment.

Medical and Surgical.

II. ORBITAL CELLULITIS.

A. Definition.

An inflammation of the cellular tissues of the orbit, usually ending in suppuration.

B. Etiology.

Septicemia, erysipelas, injuries, idiopathic and extension from adjacent sinuses.

C. Symptoms.

Constitutional symptoms may be present. Swelling of the lids, chemosis, exophthalmos. Panophthalmitis and meningitis may occur.

D. Treatment.

Medical and surgical.

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III. TUMORS OF THE ORBIT.

Carcinoma, cyst, aneurism, angioma, osteoma and sarcoma have been known to occur.

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CHAPTER XIX.

DISEASES OF THE EYEBALL.

I. EXOPHTHALMOS.

A. Synonym.
Proptosis.

B. Definition.
Protrusion of the eyeball from hemorrhage, tumors, orbital inflammation and exophthalmic goiter.

II. PULSATING EXOPHTHALMOS.

A. Definition.
Protrusion of the eyeball with pulsation of it and neighboring parts. A bruit (murmur) is heard above the eye. Usually due to an injury that causes a communication between the internal carotid artery and cavernous sinus.

B. Treatment.
Surgical.

III. EXOPHTHALMIC GOITER.

A. Synonyms.
Grave's Disease; Basedow's disease.

B. Definition.
A protrusion of the eyeballs, accompanied by rapid heart action and enlarged thyroid gland. It is a nervous disease.

C. Symptoms.
Widened palpebral fissure (Dalrymple's sign), and infrequent winking (Stellwag's sign). When eyes are turned downward, the upper lid does not follow (Graefe's sign). In severe cases the cornea is affected, due to exposure.

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D. Treatment.
Medical and surgical.

IV. MISCELLANEOUS.

A. Megalophthalmos.
Enlarged eyeball.

B. Microphthalmos.
Congenitally small eyeball.

C. Phthisis Bulbi.
Shrunken eyeball due to extensive inflammation.

D. Enophthalmos.
Recession of eyeball into the orbit. Rare and usually due to injury.

E. Anophthalmos.
Absence of the eyeball.

F. Buphthalmos.

1. Synonyms.
Hydrophthalmos; Keratoglobus; Congenital glaucoma.

2. Definition.
Progressive enlargement of whole eyeball. Increased tension. Begins before or right after birth.

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CHAPTER XX.

GLAUCOMA.

I. GENERAL.

A. Definition.

A disease characterized by increased intra-ocular tension and degenerative changes.

B. Varieties.

Acute inflammatory glaucoma, chronic inflammatory glaucoma, simple glaucoma and secondary glaucoma.

C. Etiology.

Predisposing causes are:- age (over forty), high arterial tension, arteriosclerosis and hyperopia. The exciting causes are physical and mental depression, insomnia, mydriatics, etc.

D. Pathology.

Interference of the current of the aqueous through the pupil, anterior chamber, pectinate ligament and into Schlemm's canal. There may be a blocking up of the iris angle or of Schlemm's canal, cutting off the outflow. This is followed by increased intra-ocular tension. This is the theory upon which glaucoma is explained.

E. Prodromal Symptoms.

Failure of accommodation, shown by need of stronger glasses. There may be attacks of blurred vision and halos around lights. This occurs for a year or two before the first attack.

F. Symptoms.

Ringlike

Severe pain occurring suddenly in the head and eye. Rise in temperature, nausea and vomiting. Lids swollen, eyeball congested, cornea steamy, with anesthesia of its surface, anterior chamber shallow,

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pupil dilated and oval in shape, and iris discolored. Media cloudy interfering with a view of the fundus. Vision rapidly (in a few hours) diminishes in many cases to perception of light. Tension very high. The attack lasts from a few hours to a few days. The symptoms then gradually decline, but leave the vision impaired. The other eye may be affected at any time. After a few weeks or months the acute attack recurs and later is followed by subacute or chronic glaucoma.

G. Diagnosis.

See table below for differential diagnosis, which applies to acute conditions.

H. Differential Diagnosis.

Table.

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TABLE.

	Glaucoma acute	Iritis acute	Conjunctivitis acute	Keratitis acute
Age	Over 40	Any	Any	Any
Tension	Plus	Normal	Normal	Normal
Secretion	None, or watery	None, or watery	Mucopurulent	Lacrimation
Congestion	General, especially episcleral	General, especially circumcorneal	Conjunctival, especially of lids	Rose-pink. Prominent near cornea
Cornea	Cloudy and steamy	Cloudy	Clear	Cloudy or opaque
Anterior Chamber	Shallow	Unchanged	Unchanged	Unchanged
Iris	Discolored	Discolored	Unchanged	Unchanged
Pupil	Dilated, oval	Contracted, synechia	Unchanged	Unchanged
Pain	Severe, continuous	Especially at night	None	Moderate, constant, sticking
Vision	Much reduced	Somewhat reduced	Good	More or less impairment
Corneal Sensitiveness	Diminished	Normal	Normal	Increased
Photophobia	Slight	Slight	None	Severe
Vessels	Dilated, widely distributed	Straight. Not movable with conjunctiva	Superficial. Tortuous. Freely movable with conjunctiva	Straight. Network around cornea
Field of Vision	Contracted	Dimmed slowly	Normal	May be limited by opacities
Atropin and Esserin	Atropin aggravates pain. Eserin mitigates.	Atropin soothes, eserin aggravates pain	No effect	No immediate effect. Both benefit later
Conjunctiva	No thickening	Some thickening. Not easily thrown into folds	Thickened. Easily thrown into folds	No thickening.

I. Prognosis.

Fair with proper treatment.

J. Treatment.

Medical and surgical.

II. CHRONIC INFLAMMATORY GLAUCOMA.

A. Definition and Symptoms.

Follows acute form. Tension permanently increased. Pain. Enlarged scleral vessels. Shallow anterior chamber. Pupil dilated, oval and immobile. Vision lowered or destroyed.

III. ABSOLUTE GLAUCOMA.

No perception of light. Very high tension. Cataractous lens. Dilated pupil. Shallow anterior chamber.

IV. SIMPLE GLAUCOMA.

A. Synonyms.

Chronic, non-inflammatory glaucoma; Glaucoma simplex.

B. Etiology.

Age over forty. Hyperopia, high arterial tension and arterio-sclerosis.

C. Pathology.

Same as the acute form.

D. Subjective Symptoms.

Gradual decrease in vision. Halos around artificial lights. No pain. Sometimes a feeling of pressure.

E. Objective Symptoms.

No congestion. A few enlarged scleral vessels. Anterior chamber may or may not be shallow. Pupil may be normal or slightly dilated. Lens, cornea and

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vitreous clear. Optic nerve; atrophic, white or gray. "Cupped Disc" which shows a broad scleral ring, just inside of which, the nerve drops abruptly with an over-hanging margin. Vessels drop over the margin abruptly and appear again at the bottom of the cup. Pulsation of the arteries. Tension is increased, but not constantly. Concentric contraction of visual field, more pronounced on nasal side. Sometimes irregular contractions of the visual field and isolated scotomata. Visual acuity reduced.

F. Course.

Both eyes affected at the same time as a rule. Continues for a number of years. Without treatment it ends in absolute glaucoma.

G. Prognosis.

Poor.

H. Treatment.

Medical and surgical.

V. SECONDARY GLAUCOMA.

Follows swelling of the lens, intra-ocular tumors, injuries, dislocation of the lens, hemorrhages, choroiditis, retinitis and closure of the pupil.

VI. HEMORRHAGIC GLAUCOMA.

May appear after retinal hemorrhages.

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CHAPTER XXI.

SYMPATHETIC OPHTHALMIA AND IRRITATION.

I. SYMPATHETIC OPHTHALMIA.

A. Definition.

A destructive inflammation of one eye (called the sympathizing eye), transferred from the other eye (called the exciting eye), which has been subject to a similar inflammation. The condition is comparatively rare.

B. Etiology.

Children most susceptible. Due usually to a chronic plastic irido-cyclitis, in the exciting eye, produced in most cases by a perforating wound in the ciliary region. It may follow cataract operation. It may have begun as a perforating ulcer of the cornea.

C. Pathology.

There are three theories as to the transference of the inflammation.

1. Sympathizing eye, already irritated, producing disturbances in nutrition and circulation is easily involved through the optic nerve.

2. Direct transference of micro-organisms through the optic nerve and sheath.

3. Transmission of toxins or bacteria by unknown means.

D. Symptoms (Exciting Eye).

Pain and congestion. Ciliary tenderness when pressure is applied through the lid. Minus tension. Posterior synechia. Pupil may be blocked by exudate.

E. Symptoms (Sympathizing Eye).

Chronic inflammation of the uveal tract. The

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disease may begin three ways:-

1. Slight ciliary congestion. Punctate spots on Descemet's membrane. Deep cloudy anterior chamber. Slight dilatation of pupil with some synechiae. Opacities in the vitreous.

2. May begin at once as a plastic irido-cyclitis. Pain, ciliary tenderness, ciliary congestion, pupil small and blocked, vitreous opacities, band formations in vitreous, detachment of retina and shrinking of eyeball.

3. It may begin as a neuro-retinitis.

F. Course.

Appears between the third and sixth month after the original injury. It has been known to appear as early as two weeks after, and as late as twenty years after. The sympathizing eye is very often attacked during the active inflammation of the exciting eye. It may appear without warning or there may be signs of sympathetic irritation. The course is chronic in the sympathetic eye with acute outbreaks.

G. Prognosis.

Usually causes blindness. The most favorable cases are those appearing as a neuroretinitis.

H. Treatment.

Surgical.

II. SYMPATHETIC IRRITATION.

A neurosis. It appears in the sympathizing eye as lacrimation, photophobia, impaired accommodation, asthenopia, and contraction of visual fields.

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Pathology Final

I. Define: Inflammation - give stages - causes - symptoms & results?

II State 10 things to be noticed in simple inspection of Eye.

III. Describe how you would make an exam. of the eye & its appendages to determine presence or absence of a pathological condition?

IV. Write full description of Dacryocystitis?

V Define: (A) Blepharitis Marginalis
(B) Hordeolum
(C) Chalazions
(D) Entropion
(E) Ptosis.

VI. (A) Describe fully pink eye.
(B) Name various kinds of Conjunctivitis.
(C) Define: Symblepharon - Pinguecula - Pterygium

VII. Give causes, symptoms & course of Interstitial Keratitis?

VIII. (A) Describe Senile Cataract fully.
(B) Name other types of Cataracts & give location.
(C) —

IX (A) Describe Albuminuric Retinitis
(B) Give synonyms, etiology & symptoms of Optic neuritis?

X (A) Define: Glaucoma
(B) Differentiate by means of tabular arrangement between Acute Glaucoma - Acute Conjunctivitis - Acute Iritis - Acute Keratitis.

Dr. Maguire
1-27-31

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3 Spectral Colors red, green Blue, = fundamental,
also known as trichromatic color vision. colors or primary.
Dyschromatopia =
Dichromat = mixing 2 fundamental color wave leng
Monochromat = one color, namely gray or untoned
Red blindness = protanopia
Green " = deuteranopia
Blue " = tritanopia = very rare

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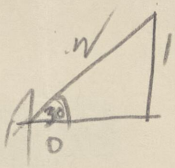

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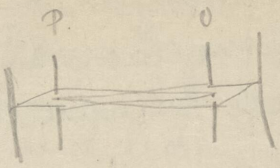

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Wm Dale Elson
Chicago, Illinois

What the Refractionist Should Know About Glaucoma

Otis Wolfe, M. D.
Marshalltown, Iowa

Glaucoma has been classed as acute and chronic, but congestive and non-congestive are terms better suited as they are descriptive.

Acute or congestive glaucoma is accompanied by pain and loss of vision, and attention is immediately directed to the eye, and suitable remedies applied. Inflammation of some degree and pain are present. It may be confused with an attack of facial neuralgia. The inflammation varies from a faint ciliary injection to a redness of pink eye, and, strange to say, it is frequently diagnosed as pink eye or iritis. It can be easily differentiated by taking the tension. The cornea is hazy and insensitive. The fundus can not be seen distinctly. The pupil is dilated or slow to contract. The anterior chamber is shallow. The well known halo may be seen around lights.

The chronic or non-congestive type, sometimes spoken of as glaucoma simplex, is seldom accompanied by pain and inflammation so that its diagnosis is frequently overlooked until great damage is done.

It is to the chronic or non-congestive type that I would especially direct your attention. It has been estimated that 30 to 40 per cent of the blindness occurring after middle life is directly or indirectly due to glaucoma—the non-inflammatory type causing the major portion. Gradual loss of vision is the chief symptom and frequently the only one. The refractionist is consequently consulted for glasses, and, therefore, he should be thoroughly familiar with the many phases of glaucoma.

The ancients did not differentiate glaucoma from cataract. Hippocrates used the word glaucoma but probably referred to cataract. The early Roman writers described a light blue or sea green pupil that accompanied certain types of blindness.

In the seventeenth century the diagnosis was somewhat clearified when Rolfinch demonstrated that, in cataract, the crystaline lens became opaque.

Mueller and Von Graefe were the first to demonstrate the increase of tension in glaucoma. The invention of the ophthalmoscope enabled the eye physician to study the fundus and recognize the non-inflammatory types.

Von Graefe was the first eye surgeon to relieve the tension and symptoms by iridectomy. Our advances since then have largely been refinements of these techniques.

It is passing strange, however, that even in our day that incipient cataract and non-inflammatory glaucoma are most frequently confused. When the two conditions exist together, which they frequently do, it requires the most exacting examinations and the most careful analysis to advise the patient so that his vision is conserved.

Secondary glaucoma, caused by a swelling of the beginning or over-ripe cataract, is frequently met. It can be readily seen how this occurs. The swollen lens narrows the anterior chamber and mechanically blocks the filtration angle. For this reason the tension should be taken in all cases of cataract.

Luther Peter says, "In view of the danger of secondary glaucoma developing in mature and Morgagnian cataracts and in view of the unsatisfactory results, which are apt to follow the extraction of such cataract if the lens capsule is ruptured, it seems imperative that, unless there is a well founded reason why the lens should not be extracted, the responsibility rests with the surgeon to advise extraction of a mature cataract, even though vision in the fellow eye is good."

Where increased tension exists, it is advisable to operate early for cataract.

Even though it is not entirely ripe, it should be removed, and the Barraquer-Green method is the operation of choice, as there is less chance of rupturing the capsule. Where non-surgical treatment for incipient cataract is instituted, the amount of secondary glaucoma present will largely determine how much can be accomplished. Miotics must be combined with cataract treatment, and unless both conditions are favorably influenced by such treatment, an iridectomy should be performed. This may entirely relieve the increased tension, and if so, the cataract treatment may be continued. If the

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tension does not remain within normal limits, the lens should be removed at the earliest possible time. The most exacting study and analysis is required to intelligently advise the patient when the two conditions exist together.

Arnold Knapp and Luther Peter both have demonstrated the frequency with which glaucoma occurs after cataract extraction. Arnold Knapp has shown from his actual clinical experience of fifteen years that glaucoma secondary to cataract extraction has not occurred where the capsule was removed intact. This is also true of secondary cataract and iritis. His experience coincides with our own. We have never had glaucoma or loss of vision occur from any cause whatever after the cataract has been removed intracapsularly. It is entirely consistent then to reason that some form of intracapsular extraction should be employed. The Barraquer-Green operation combined with preliminary iridectomy is, therefore, ideal. We employ it routinely. An eye that shows a tendency to glaucoma before cataract extraction is more apt to develop it afterward. It should, therefore, be handled by a method that is not likely to exaggerate or to precipitate the glaucoma.

S. D. Risley, in speaking of the etiology of glaucoma says: "Glaucoma is a disease, coming on at an age when wear and tear, harassing vicissitudes, misfortunes, exposures, overwork and vicious living have sapped the physiologic foundation of life; when infections have found entrance to the structure of the organism through the doorway of the epithelium; when a variety of toxic, auto-intoxic and other influences have set up vascular and cardio-vascular diseases, associated nephritis, uveitis, high blood pressure, etc. Glaucoma, in fact, rarely occurs in individuals in good health. Glaucoma is therefore a syndrome complex, the chief feature of which is increased intra-ocular pressure."

Priestly Smith has shown that the lens and the ciliary processes grow larger with advancing age while the globe remains the same. The lens and ciliary process are pushed forward against the iris root. This impedes or blocks the filtration angle. The outflow of liquids through the spaces in the pectinate ligament is mechanically interfered with. Beginning senility may cause a sclerosis of the fibers of the pectinate ligament.

SYMPTOMS.

Patients with premature presbyopia, subnormal accommodation or a deep physiological cupping may possibly be predisposed to glaucoma. Diminished light sense may be a prodromal symptom. The patient complaining of difficult vision when changing from a light to a dark room.

Cupping of the disc is a cardinal sign of glaucoma, but it often comes late. If the disc shows the slightest depression at the margin or has a greyish appearance, it should be considered suspicious. If it is a physiologic cupping, the disc margin will not be below the level of the surrounding retina. If the disc vessels bend or dip sharply at the margin of the disc, the case should be examined very carefully for other symptoms.

Glaucoma is more frequent in hyperopia, but it should be remembered that it also occurs in myopia. The patient thinks his glasses need changing, particularly those for near work. He frequently goes from one refractionist to another, feeling that his glasses are not correct or have not lasted him like they should. He will tell you "they need changing every few months." He may complain of a slight neuralgia or may present no symptoms whatever, except failing vision. The refractionist may put it down as an axiom that "whenever the patient requires a frequent change of glasses, the corrected vision gradually failing, a fundus that can be plainly viewed, that non-congestive glaucoma should be suspicioned." A slow or dilated pupil along with a shallow anterior chamber may also be noted. Inspecting the pupil by magnification and oblique illumination will greatly facilitate this.

In advanced cases the eye ball will be found firm and hard to finger palpation and the cupped disc will be seen. The diagnosis is then easy, but irreparable damage has been done. We must learn to diagnose the condition in the early state, so that preventative measures can be instituted before a considerable amount of vision is lost. We have two positive means of revealing this information. First, the testing of tension by the tonometer. Second, study of the visual fields, especially the color field.

First, before the tonometer was available, it was necessary to rely on finger palpation.

The tonometer has given us an instrument of precision. Careful and repeated tests by the tonometer will reveal an increase of tension. If it cannot be positively determined, the instillation of some of the less active mydriatics will often reveal it (homatropin). This, of course, should be followed by miotics like eserine to counteract it. Where cycloplegia is used for refraction, it should always be followed by a miotic. Eserine preferred.

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Duane says, "An eye which develops glaucoma under cycloplegia is going to develop glaucoma anyway. It is not an unmixed evil to have it develop under our eyes." I quite agree with him. We can then institute preventative measures for this insidious condition which might have otherwise escaped our attention.

Second: The charting of the fields, if frequently and carefully performed, will furnish us with our most exacting index to the progress of the disease. The color fields are most important, particularly red and green.

Briefly, the tangent screen or any type of a perimeter can be used. The hand instrument of L. C. Peter's is very efficient if his methods are followed. The Ferre Rand is the best for monocular testing. The Lloyd stereo-campimeter is excellent for cases with binocular single vision.

The important thing is the frequent and careful taking of the fields on the same instrument by the same operator so that a comparison can be made. In this manner, slight contractions of the fields will be noted. Contraction is first noted on the nasal side of the field. Central vision often remains good where there is an extensive loss of peripheral vision. Its impairment is a late symptom.

Scotomas will be noted and as the disease progresses, these will be noted to merge with the blind spot of Mariotte. Bjerrums and Seidels signs may be found by the careful technician. These will at first be relative and then become absolute. Their progress and rapidity of development will be governed by the extent of the intra-ocular pressure. If the increased tension continues, the fields will narrow (the Ronne step sign may be found), the scotomas increase and total blindness will result. The increased intra-ocular pressure is undoubtedly the cause of the progress of the condition.

Treatment

The calibre of an ophthalmologist can well be gauged by his management of non-inflammatory glaucoma. Nowhere in the realm of medicine is more ingenuity required to control the disease and the patient. It is difficult to impress on the patient the necessity of intensive study of the condition. This is particularly true in its early stages before much loss of vision has occurred. No set rules can be formulated. Each case must be studied on its merits and treated accordingly.

As the tension increases there will be a proportionate loss of field and color function. Therefore, of first importance, must be placed the immediate control of the intra-ocular tension. The question then arises, shall this be done by medicine or surgery. The best and safest ophthalmologist is he who is governed by the indications and resorts to both as indicated. Some of the best students of glaucoma have said that "sooner or later surgery will have to be resorted to, why not as well first as last?" This argument is well put and entirely logical. On the other hand, many cases are carried on safely for years by means of miotics and general treatment.

It can be safely said, however, that this is not a wise thing to do unless the patient is under the immediate attention of the ophthalmologist. The patient may otherwise be lulled into a position of false security. An occasional test for tension or charting of the field may be very misleading. If the patient is under strict observation, it is probably safe to defer operation as long as miotics hold the tension down and the fields do not narrow. The normal tension varies from 15 to 26 m. m. (Shiotz Scale) and even 30 may be normal.

If, however, repeated tests and comparisons show it at the normal limit, it should be viewed with suspicion. If the fields show any tendency to contract or the scotomas to increase, it can be regarded as abnormal and must be dealt with. A more or less continuous, though slight, increase in tension can cause great loss of vision. This low grade type is exceedingly dangerous because it is so frequently overlooked. Gradle has shown that active massage will lower the tension in a normal eye but will not do so in glaucoma.

If miotics do not positively control the tension, surgery must be combined with the treatment. Even though surgery is resorted to, it is just as important that the study and intensive examinations be continued. This is difficult for the patient to grasp. The patient expects some definite result like that following a cataract operation and is disappointed to find himself advised to continue under the prolonged observation of the ophthalmologist. The greatest tact and diplomacy are required to make the patient recognize this fact. Treatment of the eye alone is not sufficient. I quoted Risley early in this paper as stating that "glaucoma is a syndrom complex with increased intra-ocular tension" as the chief feature.

The ophthalmologist must be also a physician or call in the internist to cooperate with him. The eye condition cannot be treated as an entity. It is dependent on certain general conditions for its resulting ocular manifestations. The oph-

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thalmologist who does not so view his patient will fail in many cases that might otherwise be helped.

Briefly, I will mention the importance of focal infection, endocrine disturbances, arteriosclerosis, nephritis, diabetes, etc. The greatest advances in the treatment of glaucoma to be made in the future will probably be made by the internist and the ophthalmologist combined. It is becoming more or less definitely established that certain deficiencies of the endocrine glands are definitely associated with glaucoma. The glandular condition, however, may in turn be caused by toxemia and a source of focal infection.

Vasomotor and nervous disturbances are known to be factors in glaucoma. Worry and anxiety have been demonstrated as causing a rise in tension. To avoid these and still impress on the patient the seriousness of the condition is a problem for the refractionist.

Operation

When it has been decided to resort to surgery, there arises the question of which operation to select. This question must be answered by taking into consideration the factors governing the case. If a patient is not situated where he can be observed, the surgeon will probably wish to obtain good external drainage and be reasonably certain the tension will stay down. Here the Elliott operation would be selected. Severe cases with high tension present indications for positive surgery, i. e., external filtration of some type.

Briefly it may be stated that iridectomy is to be chosen in cases of acute glaucoma. If the tension is very high, it should be preceded by posterior sclerotomy.

Iridectomy should be performed in secondary glaucoma due to the swelling of the lens. It will also facilitate its extraction in the capsule.

In chronic non-congestive glaucoma iridotasis, or the Elliott trephine operation should be selected. Iridotasis is less severe, less traumatizing and more easily performed. There is also less chance of complication.

W. H. Wilder and others have reported some excellent results with it. It may be done first, reserving the Elliott trephine operation or other external filtration operation for the more stubborn cases.

Conclusion

It is, therefore, absolutely necessary that the one who refracts be familiar with the signs and symptoms of glaucoma. This is especially true of non-inflammatory glaucoma. Even the specialist must be ever on his guard that he does not overlook the incipient cases. He should encounter no difficulty if his examinations are thorough. He should even find and remedy many pre-glaucomatous conditions. This is a day of preventative medicine.

The patient usually considers his asthenopic symptoms as caused by a need of glasses or a change from the ones he is wearing. It is very important that the accompanying error of refraction be corrected by adequate lenses. The refractionist must accept the responsibility of separating the symptoms that are due to refractive errors and those due to non-inflammatory glaucoma. He most frequently sees the patient before considerable loss of vision has occurred. The patient presumes the refractionist is competent to advise him about his eyes and to remedy any visual defect. The responsibility, therefore, rests squarely on the shoulders of the refractionist and cannot be shifted. He must so advise his patients that vision will be conserved.

The National Society for the Prevention of Blindness has instituted an active campaign on glaucoma as a cause of blindness. They have published an interesting booklet that should be in the hands of the refractionist so that he may in turn show it to his patients.

If the refractionist is not capable of making the most exacting analysis of suspicious cases, it is absolutely imperative that he should advise the patients to consult an ophthalmologist who is. In this manner, he will be a prime factor in the great work of conservation of vision.

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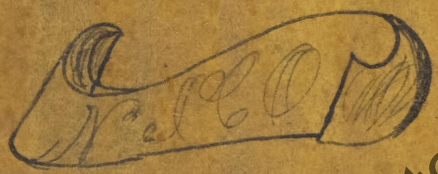

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